## REMARKS

Claims 1-14 were present in the application as filed. Claims 4-14 are cancelled above; claims 1-3 are currently pending in the application.

Independent claim 1, as amended above, is directed to a method for minimizing scarring and/or preventing excessive scar formation at an injury site. The method comprises applying to the injury site a first aid bandaging material that has been coated with a therapeutically effective amount of a defibrinogenating agent, such as ancrod. It is clear from the specification that fibrinogen and fibrin play key roles in scar formation and that depletion of fibrin is effected by administration of ancrod as a strategy for minimizing scarring. The claimed method, therefore, requires administration of a defibrinogenating agent to achieve *fibrin depletion* to prevent scarring at the wound site.

## Rejection under 35 U.S.C. §103(a)

Claims 1-14 were rejected under U.S.C. §103(a) as allegedly being unpatentable over Edwardson et al. (U.S. Patent No. 5,763,411) in view of Chen et al. (U.S. Patent No. 6,761,903) and Akassoglou et al. (cited on PTO-1449 by Applicant.)

According to the Office Action, it would have been obvious to one of skill in the art to replace the fibrin monomer coating as disclosed by Edwardson et al. with coating agents as disclosed by Chen et al. in order to provide a method to prevent scarring at injury sites as suggested by Akassoglou et al. Applicant disagrees.

The present application claims the priority of US provisional application 60/464,229 filed April 21, 2003. As a preliminary matter, therefore, the publication date for Akassoglou et al. is later than the earliest priority date of the present application and Akassoglou et al. is ineligible as prior art against the present claims.

In any event, Akassoglou et al. relates to the role of fibrin during inflammatory demyelination in the CNS and the possible significance of that role in multiple sclerosis. Specifically, Akassoglou et al. found that fibrin deposition coincided with areas of demyelination and axonal damage. Use of ancrod to deplete fibrin delayed the onset of

inflammatory demyelination. The studies by Akassoglou et al., therefore, represent the first evidence that fibrin deposition is involved in demyelination. There is, however, no teaching or suggestion in Akassoglou et al. with respect to a mechanistic relationship between demyelination in nerve and scarring. Thus, any teaching or suggestion regarding the potential benefit of administering ancrod to a wound site to prevent scarring is absent from Akassoglou et al.

Edwardson et al. teach the preparation of a concentrated fibrin preparation for use as a fibrin sealant. The fibrin composition taught by Edwardson is generated by exposing a source of fibrinogen, for example, whole blood, plasma or recombinant fibrinogen, to thrombin or a thrombin-like enzyme such as ancrod. The fibrinogen is converted by the enzyme to a fibrin monomer/noncrosslinked fibrin that is capable of being polymerized/crosslinked. Edwardson et al. also discloses that 1) in addition to the conversion step, the recovery of the noncrosslinked fibrin from serum concentrates the fibrin (col. 5, lines 34-36.), and 2) some residual level of prothrombin, factor XIII and other substances necessary for crosslinking/clotting must be present in the isolated fibrin preparation to avoid having to add exogenous material (col. 7, lines 43-47).

The objective of Edwardson et al., therefore, is to enhance clotting by applying a concentrated amount of fibrin to the wound site and subsequently facilitating its crosslinking to form a clot. The object of the present invention, on the other hand, is an attempt to prevent coagulation at the wound site, in order to reduce scarring. Ancrod is a defibrinogenating agent; defribrinogenation of blood results in an anticoagulant effect, hence, its desirabililty in treatment of stroke and other clotting disorders.

Additionally, as discussed in a previous response, Edwardson et al. is clear that fibrinogen be exposed in such a way (i.e., immobilization of ancrod or other thrombin-like enzyme on a solid support) so that the final fibrin preparation is not contaminated with enzyme, most likely because the presence of ancrod will impede clotting rather than facilitate it.

Chen et al. relates to drug delivery and in particular a drug delivery composition that enhances solubility of therapeutic agents, including defibrinogenating agents; the drug delivery composition forms a clear aqueous dispersion upon mixing with an aqueous medium, making it possible to apply a "coating" to a bandage material. There is, however, no teaching or suggestion in Chen et al. that administration of defibrinogenating agent to an injury is efficacious for minimizing scarring at the injury site. Therefore, Chen et al. also fails to compensate for the deficiency in the teachings of Akassoglou et al. and Edwardson et al. In the absence of a teaching that ancrod reduces scarring at a wound site, there is no apparent reason why one of skill in the art would modify the teachings of Edwardson et al. by substituting a coating material of Chen et al. containing ancrod, particularly where, as here, substitution, or even addition of a defibrinogenating agent with a concentrated fibrin composition capable of clot formation would obtain an opposite result, i.e., reduced clotting.

Withdrawal of the rejection under 35 U.S.C. §103(a) is respectfully requested.

It is believed that the application is in condition for allowance, and such action is respectfully requested. If a telephone conference would be of assistance in advancing the prosecution of the subject application, the Examiner is invited to telephone applicant's undersigned attorney at the number provided below.

Respectfully submitted,

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